

Reference

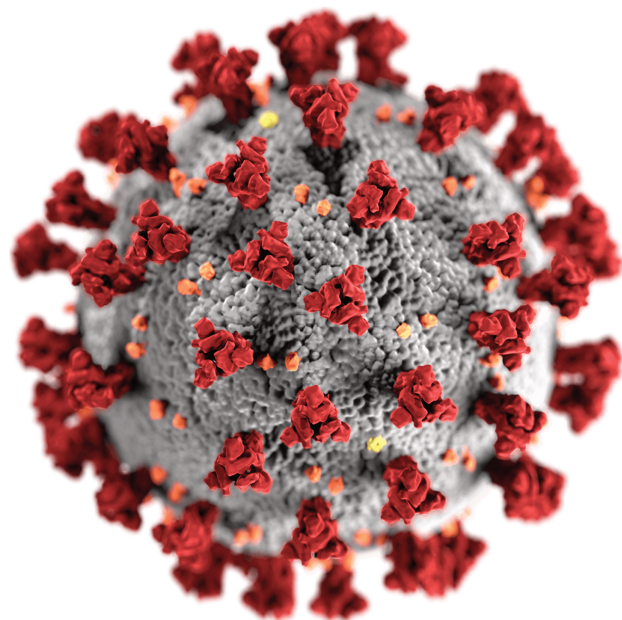
- 1 House SA, Gadomski AM, Ralston SL. Evaluating the placebo status of nebulized normal saline in patients with acute viral bronchiolitis: A systematic review and meta-analysis. *JAMA Pediatr.* 2020; **174**: 250–9.

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SARS-CoV-2 not made in a laboratory

There has been much speculation but a spectacular lack of evidence that SARS-CoV-2 originated from a laboratory. Researchers compared the genome of SARS-CoV-2 with those of the other six coronaviruses known to infect humans: SARS-CoV and MERS-CoV, which like SARS-CoV-2 can cause severe disease, and HKU1, NL63, OC43 and 229E which generally cause mild disease.¹ SARS-CoV-2 is (i) optimised for binding to the human ACE2 receptor; and (ii) its spike protein has a unique polybasic cleavage site which facilitates cell entry and led to the predicted acquisition of three O-linked glycans that provide immune evasion. Genetic data show SARS-CoV-2 is not derived from any previously used virus backbone. Although bat SARS-CoV-like coronaviruses have been grown in cell culture and animal models for research world-wide, and despite some documented laboratory escape of such viruses, genetic analysis does not support that SARS-CoV-2 resulted from inadvertent laboratory release. The receptor-binding domain in the spike protein of SARS-CoV-2 closely resembles the receptor-binding domain in known pangolin coronaviruses, which makes them a likely



origin. The origin of the unique cleavage site has not been discovered, but is expected to have arisen in a wild-type coronavirus and argues against culture-based scenarios. The authors argue that all the available evidences indicate that SARS-CoV-2 arose by natural selection, either in an animal host before zoonotic transfer or in humans following initial cryptic zoonotic transfer. What is clear is that SARS-CoV-2 is not a laboratory construct or a purposefully manipulated virus.

Reference

- 1 Anderson KG, Rambaut A, Lipkin WI, Holmes EC, Garry RF. The proximal origin of SARS-CoV-2. *Nature Med.* 2020; **26**: 450–2.

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Neonates and COVID-19

The risk of perinatal transmission from breastfeeding mothers infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is unknown. Two case reports of isolation of SARS-CoV-2 from amniotic fluid and placenta, and two cases when SARS-CoV-2 was detected in the nasopharynx of neonates ≤48 h old suggest congenital infection may occur, albeit rarely. An observational cohort study from three New York City hospitals found that 116 (8%) of 1481 mothers were positive for SARS-CoV-2 at delivery.¹ Mothers could practice skin-to-skin care and breastfeed in the delivery room if they wore a surgical mask when near their baby and practiced good hand hygiene. Neonates were kept in a closed incubator in the same room as their mothers; the mothers held them to breastfeed using the same infection control precautions. All 120 neonates were tested for SARS-CoV-2 at 24 h and none was positive. Of the 82 (68%) neonates followed up to days 5–7 after birth, 68 (83%) roomed in with their mothers and 64 (78%) were breastfeeding. Nasopharyngeal polymerase chain reaction (PCR) testing for SARS-CoV-2 was performed on 79 of the 82 neonates at 5–7 days and 72 at 14 days: all were negative. No neonates had respiratory symptoms or other symptoms suggestive of COVID-19. These data are reassuring that appropriate infection control measures are effective in preventing mother-to-infant transmission of SARS-CoV-2.

Reference

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